117 The Funduscopic Examination

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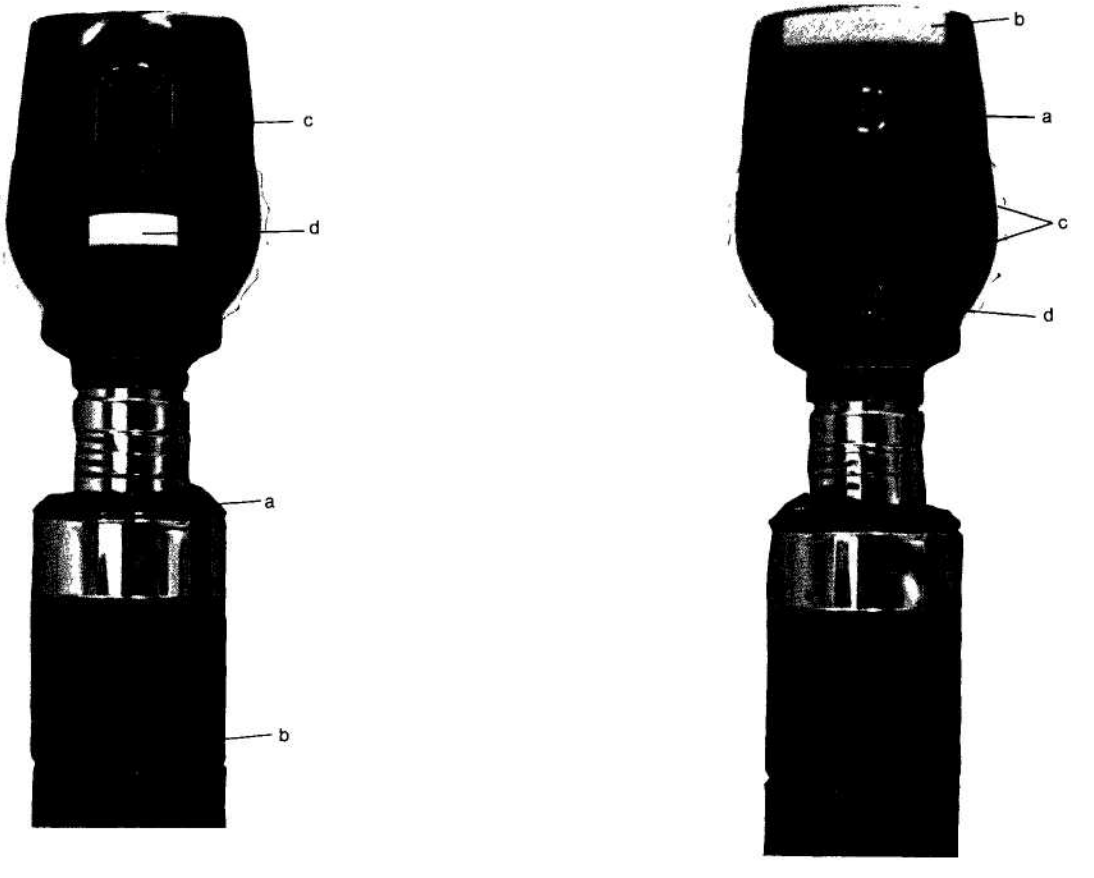
Definitions

Funduscopic examination is a routine part of every doctor's examination of the eye, not just the ophthalmologist's. It consists exclusively of inspection. One looks through the *ophthalmoscope* (Figure 117.1), which is simply a light with various optical modifications, including lenses. The ophthalmoscope illuminates the retina through the normal iris defect that is the pupil. Light rays forming the image of the retina re-emerge through the pupil. The viewing aperture (window) of the ophthalmoscope contains a lens that modifies light rays to assist the user. In the procedure, one looks at structures lying in the innermost aspect of the globe, collectively known as the *eyegrounds:* retina, retinal blood vessels, optic nerve head (disk), and to a limited degree, subjacent choroid. The pupil is frequently dilated pharmacologically to render retinal inspection easier, and for examination of the macula. One paralyzes the pupilloconstrictor muscle of the iris with nonabsorbable, short-acting topical *parasympatholytic drugs,* resulting in a larger pupillary aperture. In comparison to the ophthalmologist, the internist, neurologist, or pediatrician concentrates particularly on funduscopic manifestations of systemic disease and less on local ocular disease. Synonyms for funduscopic



A B

Figure 117.1
The instrument. *(A)* A portable ophthalmoscope in front (patient's-eye) view. *a.* Rheostat runs along circumference of tube and controls intensity of beam output. In wall-mounted instruments, this is located instead on the fixed panel. *b.* Handle containing rechargeable battery. *c.* Light emerges here, from viewing aperture (window). *d.* Equatorial wheel selects beam color, shape, and grid. *(B)* Working (physician's-eye) view of instrument *a.* Viewing aperture (window; peephole). *b.* Bumper ridge, which touches examiner's forehead. *c.* Lens wheel. *d.* Lens indicator shows which lens is in place. −2 (red 2) diopters lets the author, a myope, focus well on normal fundi from a comfortable viewing distance.

examination include funduscopy, ophthalmoscopy, and direct ophthalmoscopy. Only ophthalmologists perform retinoscopy and indirect ophthalmoscopy, which require other equipment and provide different information.

The term *temporal* is used in describing ophthalmoscopic landmarks and findings, rather than "lateral"; and *nasal* replaces "medial." The optic nerve head or *disk* is seen when one looks through the pupil from an angle about 15 degrees temporal to the optical axis (the patient's line of sight, "straight ahead"). The disk is a yellow-pink color that stands out from the redder, browner, or more orange retina proper (see Figure 117.4D). The disk is sharply demarcated temporally and to a lesser degree nasally from the *background retina,* which is all the retina that is not disk, vessels, or macula. Frequently, a narrow crescent of stippled pigment adjoins the sides of the disk, especially the temporal side (house staff have called the author to see "lesions" that turned out to be this normal feature). The disk is slightly taller than wide. The central part of the disk is paler, and is called the *optic cup* or physiologic excavation; normally this occupies less than one-third the diameter of the disk. In glaucoma and in high myopia the cup is enlarged. The transverse diameter of the disk is a standard yardstick in fundal description, so that, for example, a lesion may be characterized as "one-half disk diameter out at two o'clock, and extending two disk diameters superiorly therefrom." Although some examiners realize that the disk is 1.5 mm wide, nobody describes a lesion as 3 mm across. Near midisk, the *central retinal artery* and *vein* emerge from the optic nerve, with which they have run forward into the orbit. Each soon bifurcates into *superior* and *inferior* branches, which run "flat," that is, parallel with the retinal surface. Beyond one disk diameter out, they are called arterioles and venules. With all retinal vessels, the artery/arteriole appears slightly smaller, and distinctly lighter, more orange-red and less purple than the vein/venule. The color difference reflects the contained blood column that is visualized: the vascular walls are transparent, and deoxygenated venous blood is darker than arterial blood. Before it crosses the disk edge, each large vessel divides into a nasal and a temporal branch. Thus the principal arteries, veins, and quadrants of any retina are the *superior temporal, inferior temporal, inferior nasal,* and *superior nasal.* The avascular, dusky area two disk diameters due temporal to the disk is the *macula.* This is the area of greatest visual acuity. Apart from this zone, the background retinal color will parallel the patient's skin and hair pigmentation, from pale in light-skinned blondes to an umber shade in the darkest black people.

Normally, the largest *veins* pulsate slightly and the arteries do not, the reverse of the situation elsewhere in the body. No hemorrhage is seen in normal fundi. Any yellow, yellow-white, gray, or black interruptions of the background retinal color pattern suggest pathologic exudate, edema, or scar. No wrinkling of the retina should be seen. Chalky whiteness or erythema of the disk is abnormal, as are indistinct disk margins. Any sharp change in elevation that renders one area out of focus with the ophthalmoscope, while the remainder of the retina remains in focus, is abnormal. Tortuous blood vessels usually bespeak pathology.

Technique

The Instrument

Spend time becoming accustomed to looking through the ophthalmoscope in a nonpressured setting. Hold the instrument with the hand ipsilateral to the examining eye; both are ipsilateral to the eye being examined: examine every left fundus with your left eye, holding the ophthalmoscope in your left hand (Figure 117.2); and every right fundus with your right eye and hand. Students with strong dominance of one eye may at first experience difficulty and anguish employing the other eye. They always gain success and comfort in time. The forefinger turns the horizontal rheostat and the vertical lens wheel. A portion of this lens wheel, containing a single lens, overlies the window at any given setting. Lenses have red numbers for negative diopter values (progressively more distant focusing with higher numbers); black numbers are positive (i.e., higher black

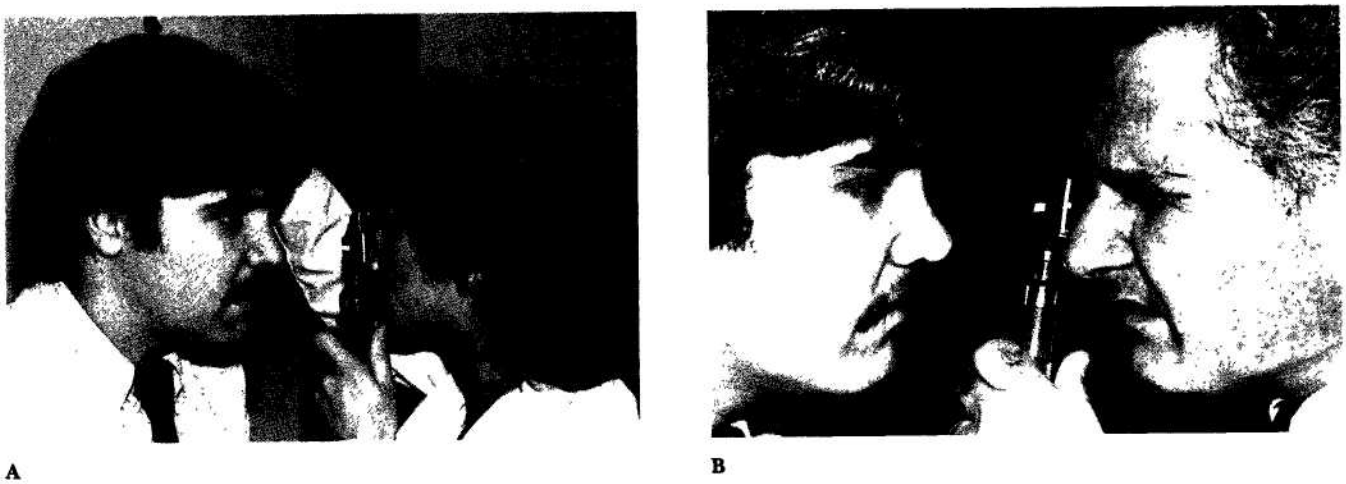


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Figure 117.2
Holding the instrument. *(A)* Examiner correctly holds the ophthalmoscope in the left hand and looks through it at subject's left eye, using her own left eye. All three organs are ipsilateral! *(B)* Bungler employs left hand and crosses over. He uses his right eye (probably because it is dominant) to try to see patient's left eye. Nose-rubbing ensues. The patient feels crowded and threatened. Visualization, and therefore diagnostic power of examination, suffers.

numbers mean shorter focal length). Of the several light beams available, only the two plain white circles are important to the generalist. The larger illuminates a wider field, but the smaller decreases corneal glare. Hold the instrument against your bony orbit, with the bumper ridge against your forehead. Practice will show how to focus it. The ophthalmoscope does not magnify images except slightly at high positive diopters. Rather, the fundus appears magnified at funduscopy because of the magnification produced by the patient's lens; aphakic fundi look tiny and far away through the ophthalmoscope. Practice turning your head and craning your neck every which way, while maintaining a constant relationship between your eye, your hand, and the instrument. A viewing aperture moved 1 mm out of your optical axis can mean loss of half the available field, so make the spatial relations as constant as though yoked.

The Procedure

Lower the room lights. Remove your and the patient's eyeglasses, but not contact lenses. Have the patient hold her glasses or put them someplace safe. Give the patient a tissue in case of tearing. Show the patient a spot directly ahead of her, on which to fix her gaze. An object or picture 1 m ahead is perfect. She will be able to fixate only with the eye that is not being examined (i.e., that is not being blocked and spotlighted by the examiner). Do not interview during ophthalmoscopy; people involuntarily turn their eyes toward a speaker, and that is the end of visualization. Hold the ophthalmoscope to your eye so that you can see well through it. Keep your other eye open, but ignore its input for now.

Begin with your light at two-thirds strength. Stand 15 degrees temporal to the patient's optical axis, your eye 30 cm from hers. Set your lens wheel at +10 diopters. Transilluminate the pupil and observe reflected red light, the red reflex. Place your contralateral palm on her forehead, with your abducted thumb on her supraorbital ridge, to prevent accidentally bumping brow or eye with the instrument. (Some doctors advocate using this thumb to help hold the eye open, but patients hate and resist it. Have a colleague try it on you if you need convincing.) Slowly move toward the patient, slowly decreasing your diopters toward zero (increasing your focal length). In this way you focus successively on cornea, lens, vitreous, and finally retina. The appearance of black spots at any point tells you that opacities are in the path of the light and will have to be accounted for or circumvented in inspecting the retina. You should wind up with the instrument only 3 to 5 cm from the patient's eye. When you see the retina, look for the first distinct structure in the area. Sometimes this will be the optic disk, more often a vessel. Bring the structure into sharp focus by rotating (changing) your lens wheel as needed. A zero reading often works well. Myopic examiners need a negative or red number (unless the patient's refractive error balances the examiner's). Aphakic patients require a high positive, often +10; by contrast, the patient who has an intraocular lens implant after cataract surgery has no such special need.

Now move along the vessel in the direction leading to larger caliber (i.e., toward "junctures" of vessels). Soon you will reach the optic disk. Study its color, its lateral margins, the size of the optic cup, the disk's elevation if any, and the pattern of vessels emerging from it. Record the details in a drawing if you wish to discuss abnormalities, to follow them over time, or to enhance your funduscopic technique. Try to appreciate pulsations of the retinal veins overlying the disk. This is easiest to see where an artery, or a bend in the vein, causes apparent diagonal interruption of one edge of the contained venous blood column.

Next, move out along the superior temporal artery, observing its normally slowly diminishing caliber, its crossovers of veins, any focal change in color, caliber, or content, and any abnormalities of background retina that come into view in the same fields. Move as far peripherally as possible, by directing the light in that direction. You will have to crane your own head and neck in an *opposite* direction, and sometimes it will feel as though you are about to twist yourself right under the table, not just 2 cm down and in! If the patient can slowly and steadily move her fixation point in the *same* direction that you want to go, that will bring more peripheral retina into view; a rapid or jerky movement by either patient or examiner will take the pupil out of the light path of your instrument. Recall, in asking the patient's assistance, that the patient's left and right are the opposite of your own! Now return to the disk by way of the vein, making parallel observations until the disk is reached. This procedure is repeated in the three other quadrants, and at the end one studies any areas of background retina that were not visualized earlier.

Finally the beam is directed temporally, or the patient is asked to look at the light. Unless the pupil is pharmacologically dilated, at best a fleeting glimpse of the macula may be obtained before pupilloconstriction or involuntary movement takes it out of view.

Do not let an interesting finding distract you from a fixed routine. Study the entity, then proceed. If you spot it out of sequence, such as with the first focus on the retina, leave it alone until its proper time. Examiners sometimes fear that they will lose the lesion and never find it again, but this never happens if one is relaxed, systematic, and dogged. Use of an inconsistent method risks lost information.

The ophthalmoscope is switched to the other hand and the other eye, and you move to a symmetric point temporal to the patient's other eye and repeat the procedure.

Put the lights back on the moment you are done. Open the shades and curtains, both for light and for morale. All other parts of history-taking and physical examination proceed better with plentiful ambient light.

Many doctors consider funduscopy the most difficult procedure in routine examination. It requires practice and persistence, but need not be an ordeal. The best results are achieved if you regularly "stack the deck" in your favor, that is, avoid the great hindrances: patient discomfort, whether physical or psychic; an uncomfortable examiner; and a struggle against physiology.

Physical and Psychological Comfort

This examination requires cooperation. Fortunately, effectiveness and humanity coincide. Assure that the patient is comfortably seated, his eyes level with your own. Your stance should be neither stooped, twisted, nor on tiptoes. If the patient is too short, prop him up on a telephone book or a cushion; if too tall, lower the bed or the examining table; if this is immobile, have the patient hunch forward to lower the torso and head (Figure 117.3). If this is not enough, direct the ambulatory patient to a low chair or stool. Using a stepstool wastes time, since the examiner needs to move to switch eyes and the patient does not.

Figure 117.3
Comfort and problems. *(A)* Examiner is too short, patient too tall, or table too high (and unadjustable). Examiner ignores discomfort in feet and writes "Fundi obscured." *(B)* Preceptor lowers patient's eyes to the level of her own, yet avoids tilt, by flexing his waist. Lesions come into view. *(C)* This patient needs distractor to hold fixed gaze. Assistant (spouse, friend, medical employee or any concerned and interested person) holds a mirror where patient can look at it with the eye that is not being blocked. *(D)* Examiner's view has just "gone blank" because the retina is not being illuminated and the funduscope is, appropriately, far out of focus for the external eye. Examiner spots this error by use of his other eye.

Supine patients lead to examiner torsion. For the patient who is paralyzed and flat, passively flex his or her waist, if possible, by raising the head of the bed to as near 90 degrees as possible. Supine examination is mandatory only when one must keep the head down as in shock, or the hips extended as in fresh iliac venous thrombosis.

All patients experience anxiety; alleviate this by informing the patient before you dim the room or shine a light in the eye. Further explanation usually creates more complications than it avoids.

Tearful patients cannot be dilated. They wash the agent out! Barring emergency, postponement of funduscopy is

wisest. The same applies when the examiner is so fatigued that visualization seems unsatisfactory or focusing impossible; while genuine pathology may be responsible for this failure, often the view is found to be crystal-clear after a night's sleep.

Do Not Fight Physiology

Ambient light causes pupilloconstriction, so lower the shades, close the curtains, and turn off the room light; but leave the shade cracked, or a small lamp, night light, or x-ray viewbox on for illumination. Otherwise, the patient may be frightened, and you may waste time inching, hunting, or stumbling in the dark. Employ the lowest rheostat setting on your instrument that shows detail and color to your satisfaction. Two-thirds of maximum often serves well. Delay study of the macula, the most light-sensitive area, until last.

The accommodation response causes pupilloconstriction, so do not have the patient fixate on a point less than 1 m away or you will counteract the relative pupillodilation of low lighting.

One looks at the retina through a hole in the iris and through four normally clear optic structures aggregately called *media*—cornea, aqueous, lens, vitreous. Total inability to visualize the retina usually means an intervening opacity, commonly a cataract or a vitreous hemorrhage. A retina that persistently looks wavy, hazy, or out of focus has the same significance. Examiner aggravation, and turning up the light to maximum, will not rectify this problem! Instead, begin the examination again, dwelling on media in detail, to localize the trouble spot. If you have not done so already, consider pharmacologic dilation, which often permits visualization via a nearby, parallel light path that is not obstructed by an opacity; for example, one can often circumvent a cataract and see the retina if the pupil is large enough.

Pharmacologic Pupillodilation

A nonsensical posture has become entrenched in some quarters, whereby an examiner who cannot see everything through a tiny miotic pupil is considered unsuccessful. This foolish expectation is never encountered among true experts: ophthalmologists refuse to render opinions unless they can see the fundus adequately, which usually means through a pharmacologically dilated pupil (Figure 117.4). Much gibberish has clouded the use of this innocuous aid. The reader should consult the Nover, Phillips, Sapira, and Schneiderman references for further information, and should seek faculty supervision for the first hundred uses of mydriatic (pupillodilating) drops.

In brief, the patient is informed about the procedure, including the need to avoid driving himself home. If he agrees to dilation, the examiner ascertains a low risk of precipitating acute narrow-angle glaucoma, by history and by lack of shadowing of the nasal half of the iris when a light is shined from the temporal side, in the plane of the iris. Baseline pupil sizes and reactions are recorded. Thereafter, one drop of 1% (or ½%) tropicamide is instilled sterilely in each conjunctival sac. The patient keeps his eyes closed for 5 minutes, to prolong contact of the agent with the conjunctiva. After 30 to 45 minutes, the pupils should be large and nonreactive. When interim inspection shows scant effect, extra drug is seldom needed, only more time. The lights are kept low for patient comfort, since some photophobia is normal after pupillodilation, for obvious reasons.

Funduscopy then proceeds in ordinary fashion, but with infinitely greater ease, speed, completeness, and accuracy. At the conclusion, the patient is reminded to report promptly any eye pain or unusual headache that day; or failure to begin regaining comfort and accommodation ("ability to read, or to focus on the television or on someone's face") within 2 hours. Any of these may mean that the ocular pressure is rising (i.e., glaucoma has been unmasked), an extreme rarity but one with great import. Any such symptom requires immediate evaluation; unless the physician is certain of a nonglaucomatous cause, immediate ophthalmologic consultation would then be requisite. For outpatients who have no glaucomatous symptoms, the only precaution is not to drive for 6 hours: mydriasis and paralyzed accommodation create road hazard even with strong sunglasses and even at night; a friend, relative, public transportation or some other means must bring them home. For inpatients, one notifies covering staff and enters a full procedure note, avoiding staff panic when Mr. Jones is found talking pleasantly but fixed and dilated! If the patient is comatose, but the need to see the eyegrounds outweighs continuous preservation of pupillary markers of neurologic status, one can affix surgical tape to the forehead, on which one has written what has been done including the time of pharmacologic intervention.

Commentary

As elementary or simply commonsensical as all these procedures may sound, many resident and attending physicians omit them, and omit pupillodilation, either through ignorance or in the false belief that they consume too much time. In fact, they save time and effort by facilitating examination and permitting better diagnosis through more accurate findings. Meticulous adherence to these procedures is an investment of effort that yields rich returns.

Problems and Solutions

When the retina "disappears" in mid-examination, look with your other eye to be sure you are illuminating the pupil; if not (Figure 117.3D), reposition the light and do not confuse the patient by apologizing. Dead batteries are another common cause of failure, but are usually heralded by brownout.

Patients who are unable to cooperate render funduscopy difficult. The intriguing proximity of the examiner's face and light creates curiosity that endures even after fears have gone. The patient fixes her gaze on examiner and light, resulting in a "moving target" of illuminated retina (and accommodation occurs, shrinking the target!). An assistant wiggling a finger in a single spot, a meter away from the patient, or an interesting picture or mark on the wall, will keep many eyes still. Any subject may wander, especially when pain or apprehension distracts him. A fixation device helps. A mirror is the simplest such device. The patient is encouraged to stare at himself in the mirror (Figure 117.3C). Lacking a mirror, one may have an assistant (or a relative, or an aide) wave and speak continuously, or even sing if speaking is not helpful enough. Some pediatricians project moving-picture cartoons onto the spot desired for fixation, with excellent results.

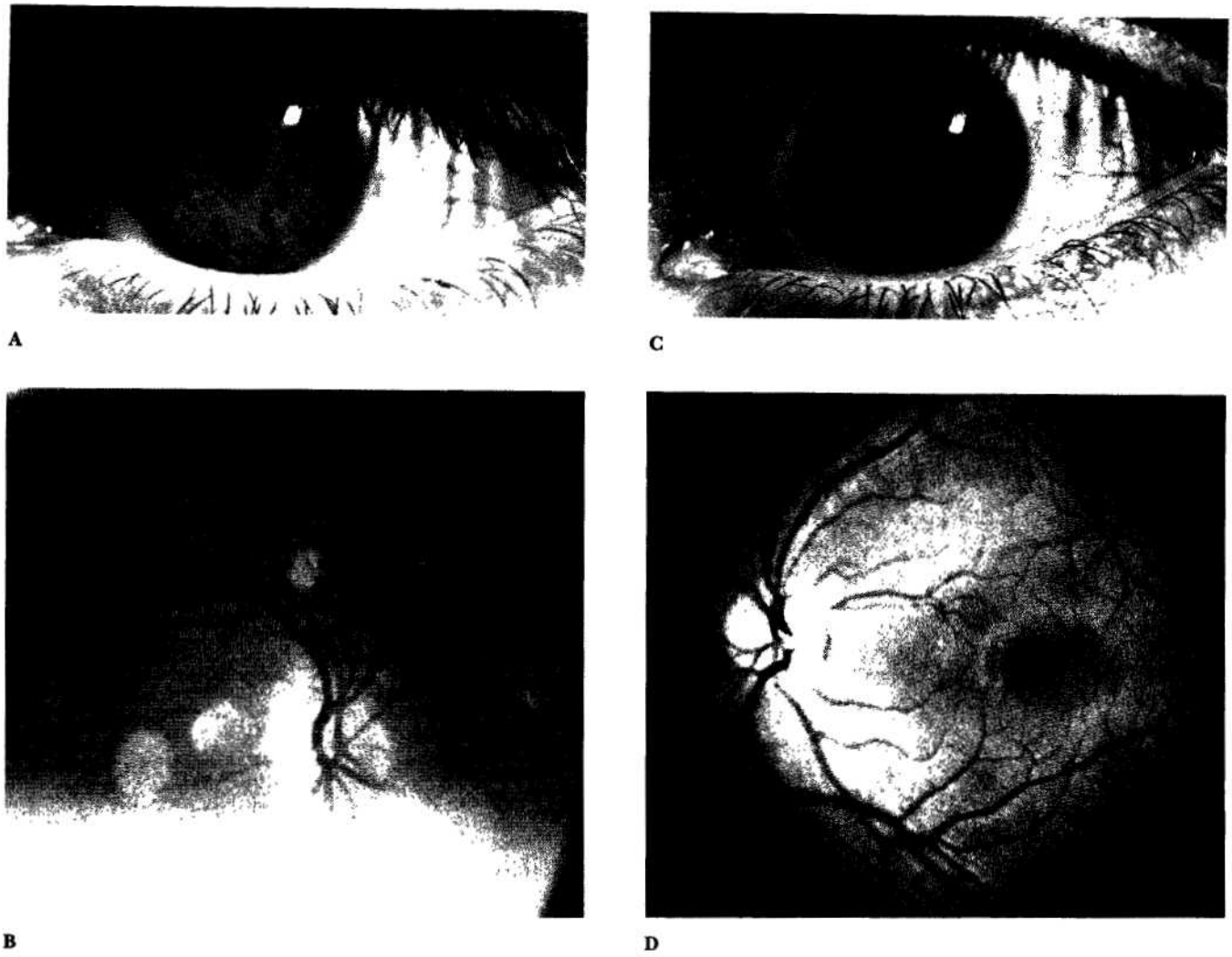

Figure 117.4
The value of pupillodilation. *(A)* External examination shows miotic pupil at baseline. *(B)* Scant fundus is revealed through this pupil. (Peripheral clouding is an artifact of photography through undilated pupil; actual funduscopy in this case showed retina only where photograph is vivid, and no image where photograph is cloudy or spotted.) *(C)* Pharmacologically dilated pupil provides much better aperture. *(D)* Retinal field of view enlarges after mydriatic treatment. Funduscopy grows easy. Almost all retinal photographs are taken through dilated pupils. The student expects a comparable view in undilated patients. The rude reality makes him wonder, unnecessarily, if his technique is defective.

If the eye reflects blinding white light back at you, so that you cannot see structures, change your angle of approach ever so slightly. This will usually cut the responsible corneal glare. A smaller beam and lower rheostat setting can also help.

Basic Science

One detail of gross anatomy may confuse the observer: a small vessel looping over the temporal disk margin, independent of the rest. This is a cilioretinal artery, a common normal variant, and not a harbinger of new vessel formation with its attendant risk of hemorrhage.

Histologic correlations at autopsy and in animal studies have provided enormous insights into ophthalmoscopic findings. We can but whet the reader's palate here. Retinal edema at first causes local elevation and a grayish loss of transparency. More chronic edema results in lipoprotein deposition, producing a characteristic sharp-edged discoloration in any of a variety of shades of yellow. This appearance is sometimes called "hard, waxy exudate" based on well-defined lateral borders and flat anteroposterior character; the term misleads, since transudates may create the same picture. It serves better to consider this a focus of chronic edema. Bear in mind the full Starling equation describing the forces leading to extravasation of fluid, and including a vascular permeability factor. Every cause of "hard exudates" includes some abnormal factor(s) from the equation. With this in mind, one can subgroup by class of cause (e.g. increased permeability, increased venous plasma hydrostatic pressure), etc., rather than memorize lists. Inflammation increases permeability in retinitis. Venous and therefore capillary pressure rises in central retinal vein occlusion, and in papilledema. Loss of pericytes allows leakage in diabetic vasculopathy. Physical damage from high pressures may act directly, and may also induce arteriolitis in systemic hypertension, creating twofold abnormality.

By contrast, so-called soft exudates, also known as cotton-wool spots, are gray-white to gray-yellow and show indistinct margins. Histologically, they represent infarction (ischemic necrosis) of the nerve fiber layer, and they should be referred to as infarcts. One can predict their occurrence in diverse vasculopathies including vasculitides. One can infer that several entities can produce both types of lesions. However, one wonders why cotton-wool spots commonly complicate AIDS (acquired immunodeficiency syndrome) in the absence of retinal infection.

Histologic configuration determines the distinctive shapes of hemorrhages in various retinal layers. For example, splinter-shaped hemorrhages must reflect extravasation in the inner nerve fiber layer. Blood will follow the path of least resistance, which is between fibers and not across them. Only in the inner nerve fiber layer do the fibers have long axes perpendicular to that of the examiner's light. Thus the leading edge of such a hemorrhage is feathery. Sapira's highly readable work summarizes the features of hemorrhage in each layer, and includes principal etiologies and import of each.

Clinical Significance

Funduscopy provides the only means for direct inspection of arteries, veins, or central nervous system in the intact, living patient. Any list of conditions in which it changes patient management and outcome would be woefully incomplete. A few selections are cited.

The finding of *papilledema,* that is, a swollen optic nerve-head usually reflecting elevated intracranial pressure, is a medical emergency. Treatment of its cause will prevent irreversible neuronal damage and somatic death. The absence of papilledema does not mean that all is well, since its development does not occur immediately after intracranial pressure rises. However, visible retinal venous pulsations exclude increased intracranial pressure, a vital datum in assessing any patient with headaches, an evolving neurologic status, or head trauma. Loss and reappearance of pulsation rapidly reflect intracranial pressure changes. Importantly, absent pulsations do not necessarily mean elevated intracranial pressure.

Retinal hemorrhages occur in diverse conditions including endocarditis, pernicious anemia, diabetes mellitus, leukemia, subarachnoid hemorrhage, and disseminated intravascular coagulation (DIC). They always deserve note in diagnosis and management; their appearance may lead to an initial diagnosis, or to assessment of progression. In severe hypertension, finding a retinal hemorrhage redefines the entity as accelerated hypertension; no given blood-pressure reading can do this. The grim prognosis of this entity, if uncontrolled, constitutes an emergency in which blood pressure control is needed not over weeks but in a day. Often this means treatment in the intensive care unit. The prevention of early death, preservation of renal function, and feasibility of subsequent outpatient maintenance reward the examiner's prowess.

The *chorioretinal lesions* of certain infections are virtually diagnostic in and of themselves. Candidal exudates mean endophthalmitis in the patient with suspected systemic candidosis. Cytomegalovirus in the patient with AIDS has a highly characteristic appearance that consists of mixed hemorrhages and yellow granular exudates, both frequently centered on vessels. A vivid if offensive *aide-memoire* for this appearance is "crumbled cheese and ketchup". Toxoplasmosis and histoplasmosis of the eye also have distinct although not pathognomonic looks on physical examination by funduscopy. Miliary tuberculosis may be confirmed by spotting choroidal tubercles.

The (apparent) complete obliteration of venous blood columns where they cross arteries, when it occurs more than two disk diameters away from the disk, is called *complete AV (arterio-venous) crossing change.* This phenomenon is seen only in vascular disease of considerable chronicity and impact. The lesion provides a permanent marker, even after normotension has been restored. Consider a normotensive man with heart failure, left ventricular hypertrophy and dilatation, and no prior medical contact. Lacking features of ischemic or valvular cause, the cardiologists speculate that chronic hypertensive damage is responsible, with cardiac dysfunction and vascular reflexes having normalized the blood pressure. (This entire scenario is common in practice.) If AV crossing changes are seen, the hypothesis is confirmed. If none are found, a primary heart muscle disorder, dilated cardiomyopathy, becomes a more likely etiology.

Proliferative *diabetic retinopathy* is a leading cause of blindness, but can be contained with laser photocoagulation. Ophthalmoscopic recognition of pre-proliferative lesions, or of frank neovascularization, will lead to measures to prevent its occurrence, and its typical sequelae of retinal and vitreous hemorrhage, and permanent blindness.

Diabetic nephropathy rarely occurs without diabetic retinopathy, so a normal retina in a nephrotic diabetic will stimulate a search for nondiabetic causes of renal disease, thus refining selective and efficient use of diagnostic resources.

In cerebrovascular disease, an ophthalmoscopic finding provides data essential for management. If the examiner sees fragments of atheroma impacted in retinal arterioles, she knows that they did not originate *in situ,* as this caliber of vessel does not experience atherogenesis. Therefore they have embolized from an ulcerated plaque in a more proximal locale, such as the aortic arch or a carotid artery. Such fragments are yellow and refractile, and typically occlude an arteriolar branch; they are called *Hollenhorst plaques.* The hazard of further embolization to the brain will strengthen the case for carotid arterial surgery. Most cerebrovascular tests measure brain texture, blood flow, or pressure; all of these may remain normal if an ulcerated plaque occurs without critical arterial stenosis; carotid arteriography is an exception, but its danger, discomfort, and expense relegate it to highly selected cases, so that the need to use funduscopy for screening is underscored.

Tortuous "sausage-link-like" retinal veins in the patient with macroglobulinemia imply marked hyperviscosity and will heighten antithrombotic efforts.

The discovery of *retinoblastoma,* a childhood malignancy that is sometimes familial, will lead to lifesaving therapy and prevention for the patient and perhaps for other family members as well. Surveillance of the remaining eye post enucleation may uncover a second primary, which afflicts many victims; if found early enough, conservative therapy may preserve vision as well as life.

Acknowledgment

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Dr. Schneiderman completed this project while a Teaching and Research Scholar of the American College of Physicians.

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